INDIRECT INJURIES OF THE OPTIC NERVE

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INDIRECT INJURIES OF THE OPTIC NERVE.

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This paper deals only with indirect injuries of the optic nerve, that is when the nerve is injured as a result of a head injury in a manner other than direct involvement by a projectile. It is based on 46 cases, 12 from an E.M.S. Head Injury centre, the remainder from the Military Hospital for Head Injuries.

Frequency of optic nerve injuries.—To estimate the frequency of this type of lesion 450 consecutive head injury admissions to the E.M.S. hospital and 1,100 to the Military Hospital were taken, amongst this group of 1,550 head injuries there were 25 cases of optic nerve injury. Ritchie Russell (1940) found 8 optic nerve injuries in a series of 600 cases. Taking these two series together the incidence of damage to the optic nerve is 1·5 per cent. of head injuries. Rowbotham (1941) gives a figure of only 0·5 per cent., but he appears to be writing of complete injuries of the nerve, while I am including the partial injuries.

To compare the frequency of involvement of the various cranial nerves the same series of 1,550 cases was used. The frequency is shown in Table I.

TABLE I.—INCIDENCE OF DAMAGE TO CRANIAL NERVES IN 1,550 HEAD INJURIES.

Olfactory	119
Optic	25
Optic Chiasm	3
Oculo-motor	15
Trochlear	15
Trigeminal (or its major branches) (excluding supra- and infraorbital involvement)	3
Abducens	15
Facial	46
Vagus	1

The 8th nerve is not included owing to the difficulty at present in distinguishing between involvement of the inner ear and of the nerve itself. It will be seen that the optic nerve is the third most frequently damaged, being exceeded only by the olfactory and the facial. It is noticeable that it is more often involved than any of the oculo-motor group.

The type of injury is shown in Table II.

TABLE II.—TYPE OF INJURY IN FORTY-SIX CASES OF OPTIC NERVE DAMAGE.

Motor-cycle accidents	17
Cycle accidents	7
Falls	6
Air-raid casualties	5
Pedestrians	4
Car and lorry accidents	5
Miscellaneous	2

The site of injury to the head is important. This could usually be determined by the presence of laceration or its scar or by the presence of localized bruising in the early stages; the presence of a closed fracture of the skull without external evidence of the site of injury has not been used in this connection. In thirty-five cases the impact was on the forehead or in the supraorbital region of the same side as the visual loss; in one patient the right optic nerve was injured when he sustained an open fracture of the left frontal region. In six cases the impact was in the region of the external angular process. In four cases no external evidence of the position of the impact was available and there was no fracture of the skull; two of these men, however, were involved in head-on motor cycle collisions so it is likely that the site of impact was frontal.

In one case only was there definite evidence of a more posterior injury, a woman who sustained an open fissured fracture in the right parietal region from falling debris during an air raid. It is possible that she was thrown forwards on her face, but as there was no external evidence of this thirty hours later, it seems reasonable that her case be accepted as an example of an optic nerve injury from a parietal impact.

This analysis of the site of injury shows the difference between these cases and those with traumatic lesions of the olfactory nerves, many of whom have occipital injuries. The *contre-coup* from an occipital injury was never observed to damage the optic nerve.

The severity of the injury is of interest. Unfortunately at present we have no absolute criterion of the severity of a head injury; a standard often used is the length of the post-traumatic amnesia. In the majority of the cases reported here this has been of some length (Table III), but there have been a few cases where it has been merely a few seconds or minutes and in one of them there was no loss of consciousness, and this has been noted by others (Thoral, 1924) in optic nerve injuries. The man with no loss of consciousness was involved in a motor-cycle skid and fell striking the left side of his forehead on the ground and sustaining much bruising round the left eye. He immediately picked himself up, and four days later when the bruising had subsided he found that he could only distinguish light from darkness with his left eye. Improvement occurred but he was left with 6/18 vision and a sector defect in the lower temporal quadrant.

This case and others of a similar type show that considerable damage can be done to the intracranial structures without the general cerebral disturbance which results in concussion, a fact equally true of the brain itself where extensive damage can be inflicted without loss of consciousness

when the impact is from an object of small size such as a bomb fragment or small piece of masonry.

TABLE III.—LENGTH OF POST-TRAUMATIC AMNESIA (P.T.A.) IN OPTIC NERVE INJURY CASES.

No loss of consciousness	1 case
P.T.A. less than 1 hour	7 cases
P.T.A. less than 24 hours	12 cases
P.T.A. 1 to 7 days	15 cases
P.T.A. over 7 days	11 cases

Course of optic nerve injuries.—In all cases in this series the effect has been immediate, and no examples of delayed blindness have been observed. Even when the nerve is only partially damaged there is often complete loss of vision in the affected eye for two to three days before recovery begins. If any useful vision is going to recover, improvement usually starts about the third or fourth day and rapidly progresses. By the fourth week after the injury all the recovery that is going to occur will usually have taken place, though one patient in this series continued to improve for about seven weeks. It is rare for subsequent deterioration to occur, but in one case which was observed for three months, vision had improved to 6/12 after four weeks, with a sector defect in the lower nasal field, while two months later vision was 6/18 and the field defect had increased to involve part of the lower temporal quadrant.

The prognosis with optic nerve injuries is worse than with injuries of other cranial nerves, except possibly the olfactory and the auditory, as the optic nerve is anatomically a part of the brain and not a peripheral nerve. In a few cases of partial injury of the nerve there is never complete loss of vision, and in one case no abnormality was noted till the patient went to the rifle-range some weeks after leaving hospital where a frontal scalp wound had been sutured.

The visual acuity following an optic nerve injury may vary from complete blindness to normal vision (see Table IV). It is possible that in some minor injuries of the nerve full fields as well as normal acuity may return, but I have not had an opportunity of following a case of this type from its earliest stages.

No cases of bilateral optic nerve injury were observed in this series.

TABLE IV.—FINAL VISUAL ACUITY IN FORTY-SIX OPTIC NERVE INJURIES.

Corrected acuity—	
No perception of light	13 cases
Less than 6/60	10 cases
6/60 and 6/36	3 cases
6/24 and 6/18	6 cases
6/12 and 6/9	12 cases
6/6	2 cases

The visual fields.—The visual field defects fall into two main groups, the first in which a scotoma is the leading feature, the second in which a peripheral sector defect is outstanding, though a certain amount of overlap

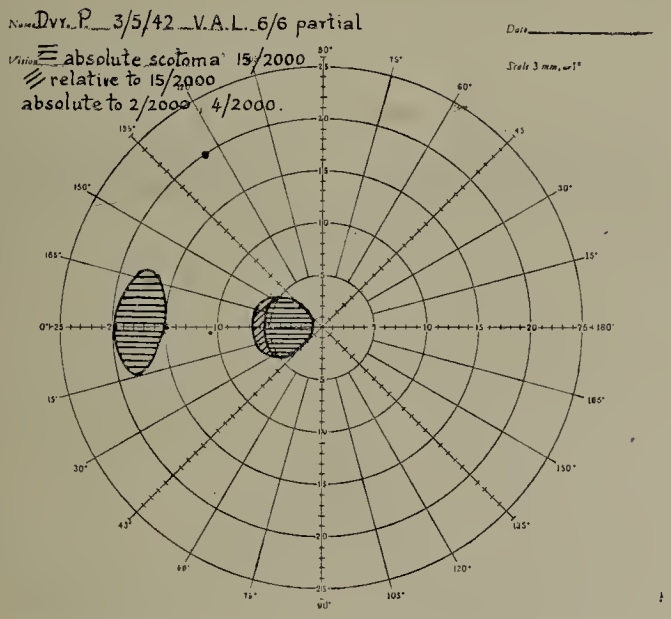

Fig. 1.—Small paracentral scotoma with V.A. 6/6 (partial).

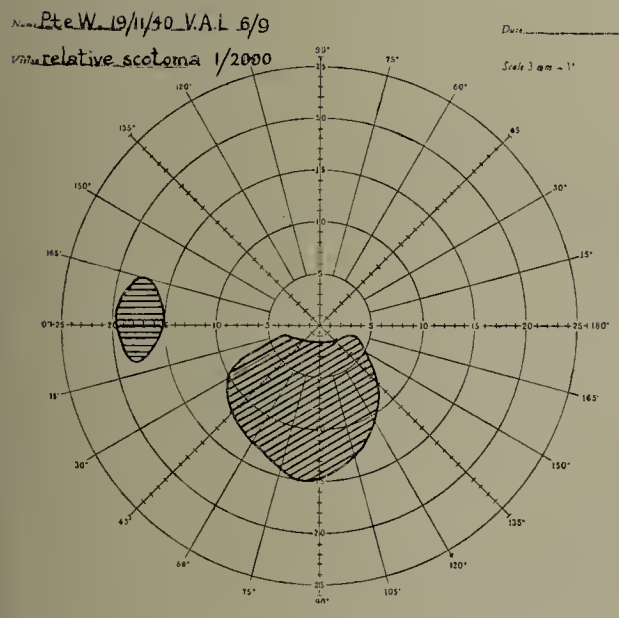

Fig. 2.

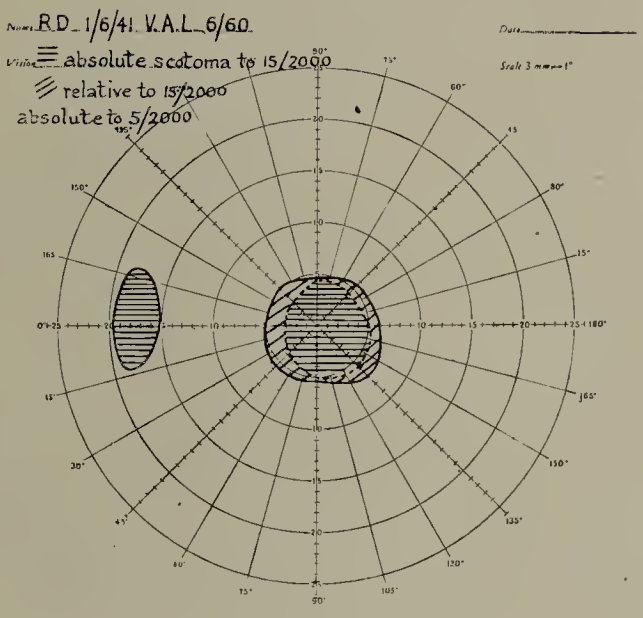

Fig. 3.

Fig. 2.—Small scotoma below fixation point. V.A. 6/9.
Fig. 3.—Dense pericentral scotoma with fading edges. V.A. 6/60.

between the two groups occurs. There were eight cases in the scotomatous group. There is no constant type of scotoma, in three cases it was pericentral, in four cases paracentral and in one case centro-cæcal.

Interesting cases are those with a small paracentral scotoma; one of these men was seen three weeks after a motor-cycle accident which had caused a post-traumatic amnesia of eighteen hours. There had been no phase of grossly impaired vision, but he complained that he could

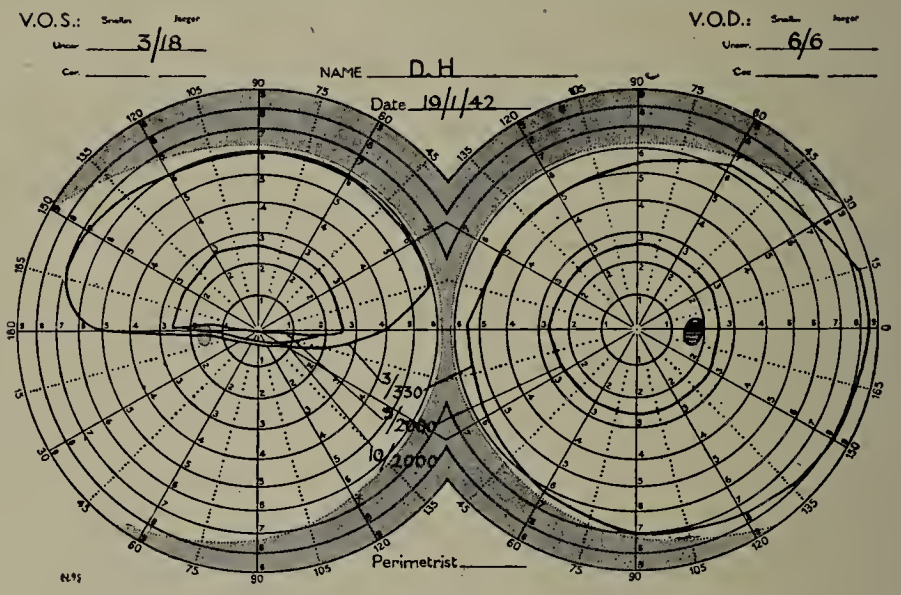

FIG. 4.—Inferior hemianopic defect. V.A. 3/18.

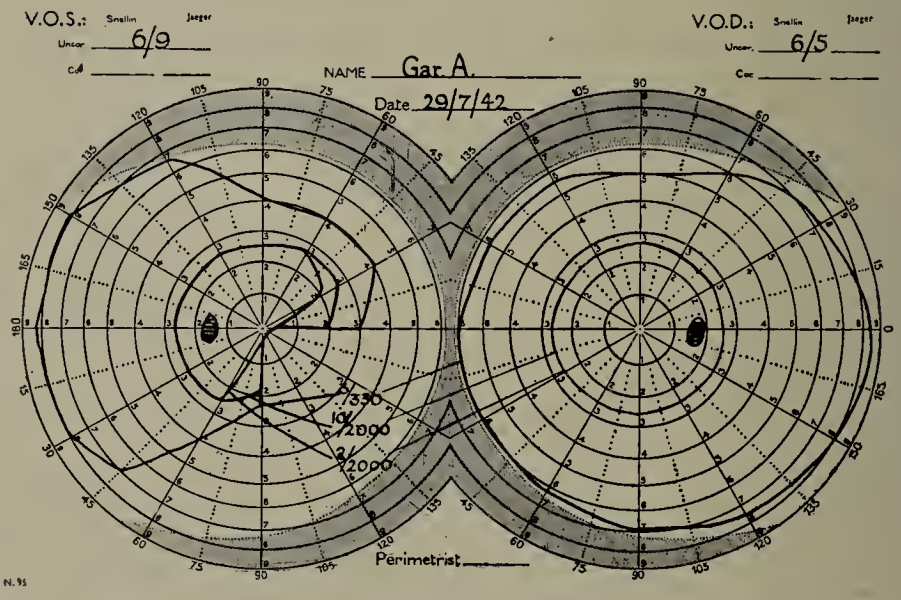

FIG. 5.—Lower nasal quadrantic defect. V.A. 6/9.

not see as well with his left eye as before the accident. On examination V.A.R. was 6/6 and V.A.L. 6/6 (partial), the fundi were normal and the pupils reacted briskly to light, there was no refractive error. It was only on careful field testing that a small absolute paracentral scotoma was

found, this case might have been diagnosed as a functional condition if screen studies had not been done. A similar case was one with a small relative scotoma to 1/2000 below the fixation point, though in this man there had at first been complete loss of vision in the affected eye. In both these cases the peripheral fields were full, in some cases with larger scotomas there have been peripheral defects in addition.

In nineteen cases the defect was predominantly in the peripheral field. These defects may be of all types from a complete temporal or inferior hemianopia in one eye to a small sector defect or general constriction with one part of the field more involved than the rest. The only type of defect which I have not encountered is an upper horizontal hemianopia. It is usual for the defects to have fading edges in both the scotomatous and the peripheral groups.

Fundus changes.—In the early stages there is no change in the optic disc; in two cases which I have examined in the first few days after the injury there have been a few scattered hæmorrhages in the retina but these were thought to be the result of direct trauma to the globe and not directly related to the optic nerve injury. Pallor of the disc in the complete injuries is usually first noticeable towards the end of the third week and then rapidly progresses to the pallor of complete optic atrophy. In the cases with slighter damage to the nerve the appearance of pallor of the disc occurs later and in some cases never becomes noticeable.

The pupils.—In severe injuries of the optic nerve the pupils are usually equal, the pupil on the affected side reacts sluggishly or not at all to direct light but reacts briskly to consensual light. In slight degrees of damage there may be no alteration in the pupillary reaction.

It should be noted that inequality of pupils does not occur unless there is a coincidental 3rd nerve lesion or a traumatic mydriasis; otherwise in an unconscious patient the finding of one pupil which does not react to direct light while the other one reacts normally may be misinterpreted as a stage in the development of the Hutchinsonian pupil associated with extradural hæmorrhage. The finding of a normal consensual reaction will also prevent this error.

One patient in this series had a traumatic Argyll Robertson pupil on the same side as the optic nerve injury, probably due to trauma to the efferent pathway of the light reflex (Nathan and Aldren Turner, 1942).

Differential diagnosis.—It is not uncommon after a head injury for a patient to complain that vision in one eye is not as good as formerly, the commonest cause of this is the unmasking of a refractive error which

had not been noticed previously. One patient, who knew that he had an amblyopic eye previously found that vision in this eye was further reduced after his injury and on testing his fields he was found to have a paracentral scotoma showing that he had had an optic nerve injury on the side of the amblyopic eye.

Hysterical amblyopia may occur in one eye following a head injury. The assertion by Terrien (1936) that immobility of the pupil to light is a constant accompaniment of an optic nerve lesion, a finding which he takes as the major distinction from an hysterical amblyopia, is, of course, unjustified in the case of partial injuries of the nerve.

Crescentic tears of the choroid, which may be followed by optic atrophy sometimes follow frontal blows and cause considerable impairment of vision, but these can be seen with the ophthalmoscope. Occasionally following a head injury a macular hæmorrhage which may be followed by macular degeneration occurs, this lesion will cause a pericentral scotoma and may be very difficult to recognize even when the pupil is dilated.

Concomitants of optic nerve injuries.—As would be expected, concomitant lesions of other cranial nerves were frequent in this series. In eleven cases there was unilateral or bilateral anosmia, in ten there was a partial or complete 3rd nerve lesion and in four a 6th nerve lesion; in only one case was there a complete sphenoidal fissure syndrome and in this man all the cranial nerves recovered except the optic and to a lesser degree the 6th. Six patients had cerebrospinal rhinorrhœa, one a subarachnoid aerocœle and two carotico cavernous aneurysms. In no case was there evidence of hypothalmic disturbance, such as may occur with injuries of the optic chiasm.

Radiology in optic nerve injuries.—Table V shows the findings in routine skull plates.

TABLE V.—EVIDENCE OF SKULL FRACTURE IN FORTY-SIX OPTIC NERVE INJURY CASES.

No fracture demonstrated	19 cases
Fissure in frontal bone	20 cases
Depressed frontal fracture	5 cases
Parietal fissure	1 case
Temporal fissure	1 case

Of greater interest are the special views of the optic foramina in these cases. Radiological demonstration of a fracture into the optic canal was first given by Goalwin in 1926 when he found a fine fissure extending into the canal. Foraminal pictures are difficult to interpret and it is

essential to have views of the foramina of both sides for comparison. In this series of forty-six cases, nine have not had these special views taken, while in thirty-three cases no abnormality of the foramina could be seen. In four cases only was an abnormality discovered. In one the optic foramen on the affected side was smaller than the opposite one, in this case the injury was eleven years earlier and the appearances were equivocal. In the second case in which there was a sphenoidal fissure syndrome in addition to the optic nerve injury, there was apparent compression of the optic canal and a fracture line extending into it. In the other two cases a fine fissure was found running into the lower part of the canal on the lateral side.

These findings indicate the rarity of radiological evidence of a fracture involving the optic canal in cases of optic nerve injury. The canal is several millimetres in length and if tomographic studies were carried out it is possible that fine fissures into it might be demonstrated in a higher proportion of cases.

Pathogenesis of optic nerve injuries.—The main difficulty in discussing this problem is the absence of post-mortem studies in cases which have been known in life to have injuries of the optic nerve. I have had no pathological material from my cases.

The optic nerve consists of three parts, the intraorbital portion being about 25 mm., the canalicular about 4 to 10 mm. and the intra-cranial 10 mm. long (Duke-Elder, 1932). The orbital portion has considerable slack in it to allow for movement of the eyeball but the canalicular portion is closely bound down especially on its superior aspect in its dural sheath which is united to the periosteum. It is practically certain that injury to the nerve takes place in the canalicular or intracranial portions owing to their immobility, and this position of the lesion corresponds with the appearance of pallor of the discs about three weeks after complete lesions.

The mechanism of these injuries originally advanced by Berlin was that a fracture into the canal involves the nerve—he based this on the post-mortem statistics of van Hoelder who stated that in 73 per cent. of fractured bases the optic foramen was involved. The criticism of these findings is that a high proportion of van Hoelder's cases were gunshot wounds, particularly suicides who had fired into their mouths, the effect being a different one from the ordinary case of fractured base.

Later post-mortem studies on fractures of the base in closed injuries, especially those of Bathe Rawling (1904) showed that the optic canal was only rarely involved, the majority of basal fractures in this vicinity tended

to pass to or from the cribriform plate between the two optic foramina or more laterally through the sphenoidal fissures. Small fissured fractures sometimes radiated to the optic foramina but they were usually of so slight a nature as to be incapable of causing any gross lesion of the nerves.

More recently X-ray studies have helped in this problem. My results show the rarity of demonstrable fractures into the optic canals in these cases; others (Barkans, 1928) who seem to assume the presence of fractures were unable to show them radiologically.

Rollet, Paufique and Levy (1930) however were able to demonstrate fractures in ten cases of optic nerve injury, but in their illustrations the fractures are difficult to be sure of and in only one of them was there a detached fragment of bone which might have compressed the nerve. Although these authors claim that all optic nerve injuries are accompanied by a fracture into the optic canal, they do not think that detachment of bone fragments sufficient to compress the nerve is of any general importance.

Some confusion of thought seems to exist on this point; a fine fissured fracture into the optic canal cannot of itself cause an optic nerve injury unless there is detached bone compressing the nerve; there must be some other mechanism involved—if a fracture is present it is an accompaniment of, and not the cause of the nerve lesion.

A second explanation is that the intracranial portion of the nerve may be compressed by a fractured anterior clinoid process, Rawling found that this type of fracture was fairly common. Pringle (1922) in an important study on the question of optic nerve injuries did post-mortems on 174 patients dying after head injuries, these were acute cases in which no clinical study of visual function could be made. He found six cases with fracture of the anterior clinoid process but in none of these did there appear to be any injury to the nerve or hæmorrhage into its sheath.

There does not appear to be any direct evidence that optic nerve injuries can be caused in this way, but there are two remarkable cases described by Lillie and Adson (1934) in which there was delayed unilateral visual failure after head injuries, one of these cases was explored and there was found to be a fracture through the anterior clinoid which had extended into the optic foramen, callus had formed which had compressed the optic nerve to two-thirds of its normal size; the callus was removed and the nerve decompressed by removal of the roof of the optic foramen; no hematoma was found in the nerve sheath. There was however no improvement in vision.

A number of authors have accepted the view that hæmorrhage into the sheath of the optic nerve with compression of the fibres is the cause of the visual failure in these cases. Pringle (1922) in his 174 post-mortems found blood in the nerve sheath in 16 cases, these cases were all unconscious from the time of the head injury till they died and no note on their visual acuity could be made. From this evidence he believed that optic nerve injuries were due to compression by blood and he carried his belief to the extent of operating on three cases of uniocular blindness following closed head injury; in all three cases he found blood under tension in the nerve sheath, but there was no subsequent return of vision in any of the cases, he ascribes this to the fact that the operations were done between two and four weeks after the injury and irreparable damage had been done to the nerves.

There seems to me to be circumstantial evidence against this view of the mechanism of optic nerve injuries. In spontaneous subarachnoid hæmorrhage from an aneurysm or an angeioma there is frequently blood in the sheath of the optic nerve and unilateral optic atrophy is not apparently a consequence of this, unless there is direct pressure on the nerve by the aneurysm. In these cases also there are frequently fundus changes, such as papillœdema and subhyaloid hæmorrhage, probably from the increased pressure in the sheath causing obstruction of the central retinal vein as it crosses the subarachnoid space round the nerve as indicated by Riddoch and Goulden (1925). Retinal changes are uncommon in the early stages of optic nerve lesions and this would appear to be against the theory of intravaginal hæmorrhage as the causative factor.

I think that the most probable explanation in the majority of cases is a vascular lesion, either hæmorrhage or thrombosis in the substance of the nerve, resulting from the injury—a theory similar to that put forward by Traquair, Dott and Ritchie Russell (1935) to account for injuries of the chiasm.

The blood supply of the optic nerve has been studied by Abbie (1938) and by Eugene Wolff (1940). Their conclusions are based on injection followed by dissection under a binocular microscope. The intracanicular portion of the nerve is supplied by small branches from the ophthalmic artery, and these contribute to a pial network of vessels which surround the nerve. From this network small arteries pass at right angles into the nerve taking with them a coat of pia and being covered by glial tissue which forms the septa which divide the nerve into a large number of separate bundles. As the vessels pass into the nerve they divide and send branches

forwards and backwards. There is however another source of blood supply for part of the intracanicular portion of the nerve. Abbie states that just after the central artery of the retina enters the optic nerve behind the globe it gives off a recurrent branch which extends posteriorly as far as the optic foramen.

There is little possibility of movement of the optic nerve in its canal and a blow on the frontal or temporal region may result in a sudden jarring of the nerve, probably causing violent impingement against the bony wall of the canal. This would be sufficient to rupture small vessels in the septa of the nerve, followed by local thrombosis and softening.

This hypothesis lacks pathological confirmation but it seems to be the most satisfactory one in the case of partial injuries at any rate, and would account for the scotomatous type of field defect which is difficult to explain on the basis of blood in the nerve sheath. It is possible that the scotomatous defects are caused when the damage is to the recurrent branch of the central retinal artery, while the more common type of hemianopia or sector defect is produced when the damage is chiefly to the vessels entering the nerve from the pial anastomosis investing it. The complete blindness immediately after the injury which may last for two or three days even in the partial injuries is comparable to the spinal shock which occurs in injuries of the spinal cord which later recover to a greater or less extent, the physiology of this remains obscure.

In the complete lesions of the optic nerve a similar vascular mechanism may apply, but in some cases there is probably actual laceration of the nerve or compression of it by detached bone fragments.

Summary.

(1) 46 cases of indirect optic nerve injuries have been studied.

(2) Attention is drawn to the partial injuries of the nerve where visual acuity may be practically normal but small scotomata are present.

(3) The site of the injury is almost invariably in the frontal region.

(4) In only four cases has any radiological abnormality of the optic foramina been demonstrated.

(5) Arguments have been put forward to support the view that the mechanism of the injuries is intraneural vascular damage.

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